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Adaptive and Genomic Explanations of Human Behaviour: Might Evolutionary Psychology Contribute to Behavioural Genomics?

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Abstract. Evolutionary psychology and behavioural genomics are both approaches to explain human behaviour from a genetic point of view. Nonetheless, thus far the development of these disciplines is anything but interdependent. This paper examines the question whether evolutionary psychology can contribute to behavioural genomics. Firstly, a possible inconsistency between the two approaches is reviewed, viz. that evolutionary psychology focuses on the universal human nature and disregards the genetic variation studied by behavioural genomics. Secondly, we will discuss the structure of biological explanations. Some philosophers rightly acknowledge that explanations do not involve laws which are exceptionless and universal. Instead, generalisations that are invariant suffice for successful explanation as long as two other stipulations are recognised: the domain within which the generalisation has no exceptions as well as the distribution of the mechanism described by the generalisation should both be specified. It is argued that evolutionary psychology can contribute to behavioural genomic explanations by accounting for these two specifications.

1. Introduction

1.1. *Evolutionary psychology and behavioural genomics*

Currently, evolutionary psychologists are attempting to unify psychology with the hard science of biology (e.g., Cosmides and Tooby 1994, 1997). Ironically, the two grand biological approaches to behaviour are themselves not unified: *evolutionary adaptive accounts* on the one hand and *behavioural genetics* and *behavioural genomics* on the other seem to be developing in quite autonomous ways without much mutual interest. There is little author overlap between the most representative journals of both disciplines and their

textbooks seem to ignore each other (Bailey 1998). The question that we will be concerned with in this paper is whether despite their differences, evolutionary psychology might to a certain extent be unified with, or contribute to behavioural genomics.

One of the reasons that evolutionary psychology and behavioural genomics may ignore each other is that both approaches, although biological in origin, have a different history and orientation towards the biology of behaviour. Evolutionary psychology in succession to sociobiology presupposes that human stable behaviour patterns and standard ways of experience are the products of the human brains and focuses on the evolved properties of the human nervous system (see e.g., Barkow et al. 1992; Buss 1995, 1999; Cartwright 2000; Cosmides and Tooby 1995; Cziko 1995; Donald 1995; Passingham 1982; Plotkin 1997; Van Hezewijk 2003; Barendregt 2003b). The human mind/brain is understood as being composed of functional parts like other bodily organs are structured. Evolutionary psychology borrows the conceptual framework of natural selection of inherited characteristics from evolutionary biology and claims that the human nervous system is functionally organised to serve survival and reproduction. These brain functions are referred to as 'psychological adaptations' which reflect the domain specific adaptations the human species has developed during evolution as solutions to problems of earlier ancestors of the species (Gigerenzer 2001). Two main theoretical tenets of evolutionary psychology can be summarised as follows. Firstly, because selection typically produces complex and species universal adaptations, evolutionary psychology disregards variation and concentrates on the *universal human nature*. Secondly, the universal human nature consists of hundreds or thousands of evolved modules, selected to perform specific functions. Thus, evolutionary psychology's explanations of stable behaviour patterns involve a focus on the universal, yet domain specific, functional design of human nature.

Behavioural genomics, on the other hand, is the next (post-genomic) step in genetic research of behaviour. It is not so much interested in the evolutionary and adaptive aspects of behaviour but much more in gene-behaviour causal pathways. Traditionally, behavioural genetics asks *whether* and *how much* behaviour is influenced by genes. Evidence for genetic influence comes from inter alia adoption and twin studies with which the *heritability* of behavioural traits can be measured (see e.g., Boomsma et al. 2002; Spector 2000). These quantitative analyses are now considered as important but only first steps; the next steps concern the mechanisms by which specific genes affect behaviour (Dick and Rose 2002; De Geus 2002; McGuffin et al. 2001; Plomin and Essi 2001). Behavioural genomics involves the analyses of the contribution of genes to behavioural functions, tracing the chain of cellular

and molecular mechanisms between behavioural and genetic levels (Plomin and Crabbe 2000). Thus, in contrast with heritability research behavioural genomics includes molecular genetic studies in order to understand how genes are expressed in specific behaviours.

1.2. *Unification*

The concept of unity is itself not a unified concept and has had many different interpretations in the history of science (see Morrison 2000, Chapter 1). In this paper we will be concerned with the unification of theories and explanations. Gold and Stoljar (1999), following Maudlin (1996), distinguish three different intertheoretic relations that may be denoted by 'unification': *dissolution*, *reduction*, and *conjunction*. Unification by dissolution refers to the situation when two theories are dissolved by a conceptual advance, which reveals that both domains are features of a single theoretical domain (e.g., in the philosophy of mind double-aspect theories attempt to achieve unification by dissolution). Theory reduction establishes a unification between two theories by showing how one theory is completely derivable from the other one (Nagel 1961: chapter 11). Finally, unification by conjunction occurs when two theories are simply joined together. Although a weak kind of unification, conjunction is not trivial (Gold and Stoljar 1999). Firstly, both theories should not be mutually inconsistent or conflicting.[1] Secondly, the relation must be somehow interesting enough. As Maudlin puts it, that a theory of embryonic development is consistent with a theory about the formation of the rings of Saturn is not sufficient to render the two unified (Maudlin 1996: 130). Thus, in order to qualify as a unification the conjunction of two theories must bring something new to them: for example, if an elliptical explanation is complemented with information provided by the other theory. Also, as suggested by Lakatos (1970) a unification is not trivial if the conjunction predicts the facts that the two theories predicted as well as some new facts that neither did seperately.

In this paper, we shall focus on the conjunctive unification of adaptive and genomic explanations. More specifically, the question is whether adaptive explanations can contribute to behavioural genomic explanations. In order to do this, we must show, firstly, that adaptive and genomic explanations are not inconsistent. One reason why they might be inconsistent is that they focus on different (perhaps even conflicting) features of behaviour: evolutionary psychology studies features of behaviour shared by all human beings, while behavioural geneticists study a population's variance. This issue will be reviewed in section 2 and we will conclude that there is nevertheless a substantial overlap between both approaches. Secondly, it must be shown that the conjunction of evolutionary and genomic explanations is not trivial.

In section 3 we will discuss the structure of biological explanations from a philosophy of science point of view, while in section 4 we will apply these insights to explanations from behavioural genomics as well as evolutionary psychology and propose two reasons why evolutionary psychology might be relevant to behavioural genomic explanations. In the final section, we will discuss some limitations of our proposals.

2. Variation and uniformity

2.1. *Human monomorphism*

Despite the theoretical connections between evolutionary psychology and behavioural genomics as reflected by the shared use of a 'gene' concept, the two approaches differ in at least one important respect. As observed above, evolutionary psychology focuses on the structure of behaviour aiming to uncover the *uniform* functional design of human nature. By doing this, the evolutionary approach aims at the qualitative characteristics and the explanation of 'normal' behaviour (Cosmides and Tooby 1997). Behavioural genomics on the other hand, investigates the genetic background of *individual differences* approaching cognition as an individual trait (Plomin 2002). Its main methodology is the analysis of *variance* trying to discover the proportion of variance of behavioural traits attributable to genetic variance. So, one difference between evolutionary psychology and behavioural genomics is the way that 'genes' are conceived: either as the information carriers for the retention of universal adaptations in the species or as the causes of differences between individual members of that species.

This difference has been put forward most explicitly by Tooby and Cosmides (1990). Their argument is that the complex functional design of human adaptations requires design *monomorphism*. The reason is that complex systems depend on the interdependence of the integrated parts. In order for the parts to interact in a predictable and organised way, each part must be functionally uniform and regular. In other words, each part must rely on the behaviour of every other component. Although component parts may show considerable variation in some properties (e.g., no two stomachs have exactly the same size and shape) the basic design of each organ is the same. So, from the perspective of the adaptive, qualitative design every individual of a species is uniform and individual variation must be minor and quantitative. For sexually reproductive organisms like humans, this constraint becomes even more pressing. During sexual reproduction, the genes of the parents are to a large extent randomly recombined, forming genetically different and unique offspring. Such a mixture of genes can only work if the functional

designs related to these different genes are extremely similar to begin with (Pinker 1994: 326).

Evolutionary psychologists do not claim that natural selection can operate without heritable variation. On the contrary, heritable variation is essential for selection processes (Hull et al. 2001). But according to Tooby and Cosmides (1990), heritable variation is essential only for the evolutionary processes as *input* for natural selection. The *output* is a monomorphic adaptive design. The variation that is initially necessary is eliminated by natural selection: the longer natural selection acts, the more heritable variation is 'used up' and the better, 'winning' variant becomes the more common one. Eventually this variant is fixed in the gene pool and becomes a universal part of the species' genetic endowment.

A corollary of this is that adaptively significant characteristics tend to have *low or zero* heritability (Crawford and Anderson 1989). Heritability as measured by behavioural genetics, which is the basis of behavioural genomic analyses, indicates the proportion of phenotypic variance attributable to genetic variance. But if selection has operated on a trait intensely for a long time, all genetic variation related to that trait must have been eliminated and, consequently, heritability reaches zero. It is claimed that the converse also holds: traits with much heritable variation are those traits that are *not adaptations*. According to Tooby and Cosmides, then, 'behavior geneticists tend to be studying phenomena that are not themselves adaptations . . . , but the raw material out of which future adaptations may someday be made' (1990: 38).

If this analysis were correct, there would be a problem for attempts to conjunctively unify evolutionary psychology and behavioural genomics. The gene-behaviour relationships genomics is interested in would be very different from the gene-adaptation relationships evolutionary psychologists focus on. There would be a 'natural' dividing line between both disciplines with on the one side pan-human adaptations and on the other side human variation of characteristics that are not adaptations.

2.2. *Adaptive genetic variation*

For a number of reasons, however, the argument of Tooby and Cosmides seems to be problematic. One problem is that genetic analyses demonstrate moderate to high heritabilities for almost all behaviours that have been studied (McGuffin et al. 2001). According to Plomin and Colledge (2001), the question is not whether there are domains in psychology which show genetic influence, but whether there are areas that do *not* show any heritability. Also, human traits that are supposed to have been strongly selected such as language ability and intelligence have rather high heritabilities and

this seems hard to reconcile with the view that adaptations have zero or low heritability (Plomin and Petrill 1997; Miller and Todd 1998).

It also seems that the interdependence of the component parts of integrated systems does not prohibit qualitative variation in those parts. David Sloan Wilson (1994) pointed to the possibility that genetic polymorphisms can be maintained by natural selection if the maintained phenotypes have a fitness that is sufficiently high while the intermediate forms have a relatively low fitness (a saddle shaped fitness function). An example of this is the escape tactics of garter snakes (Bailey 1998; Brodie 1989). Under threat mottled snakes tend to remain motionless, while striped snakes tend to flee in a straight line. Intermediate phenotypes have lower fitness (e.g., mottled snakes fleeing in a straight line), but the two phenotypes which are maintained have a fitness high enough to be selected for. Another example is the evolutionary very relevant phenotype of fertility. Human fertility contains genetic variation that may be adaptive and seems to co-vary with other traits related to fertility, like age at puberty or sexual and parenting behaviour (Rodgers et al. 2001).

Both Wilson (1994) and Buss and Greiling (1999) refer to *frequency dependent selection* as a possible cause for adaptive genetic variation. If the fitness of one variant depends on the presence of other variants, all these variants may be sustained by natural selection. An example is biological sex: the reproductive success of one sex depends on the availability of mates of the other sex and as a result selection will favour an equilibrium with equal ratios for both sexes. Frequency dependent selection also occurs within sex, for instance the parasitising mate strategies of the bluegill sunfish. It's strategy is to mate with a female, but to leave the parenting of the offspring to other males. The parasitising strategies can only be successful if they are not too frequent, because their success depends on the presence of enough non-parasites that will parent the offspring.

Frequency dependent selection may also obtain among human behaviour. The evolution of co-operation in the context of a prisoner's dilemma is a point in case (Axelrod 1984; Badcock 2000: chapter 3). When faced with a series of iterated prisoner's dilemmas actors can perform a number of different strategies. Computer simulations demonstrate that the strategy to always defect is more successful and outreproduces the strategy that is always co-operative. But even more successful is TIT FOR TAT (Axelrod 1984), the strategy that simply copies the previous move of the opponent. Interestingly, TIT FOR TAT is equally successful as the co-operative strategy, because mutually they will be constantly co-operative. As a result, in a population the co-operative strategy and TIT FOR TAT will both be maintained, thanks to TIT for TAT that functions like a buffer against defective strategies.

It is important for present purposes that – contrary to what evolutionary psychologists assert – the genes referred to by evolutionary psychology and the genes used in behavioural genomics may well be the same genes. This challenges the view that evolutionary psychology and behavioural genomics are disconnected and completely separated fields of research. The question then arises how explanations in both disciplines relate to each other if they indeed involve the same genes. Might an evolutionary perspective contribute to explanations in behavioural genomics?

3. Explanations in biology

3.1. *Laws*

Analyses of biological explanations often turn towards the matter of the existence of laws in biology. Laws are commonly interpreted as generalisations that are, firstly, exceptionless. Any case in which the antecedent of the generalisation but not the consequent holds, excludes the generalisation from being a law. Secondly, laws are not limited to specific locations or specific times, i.e., the scope of the quantifiers in a law covers all space and time. Additionally, laws must be able to support counterfactuals in order to distinguish truly lawful generalisations from generalisations that merely accidentally have the same form of laws. It is because of these characteristics that laws function in explanations. According to the classic interpretation, to explain an event is to show why it had to happen (e.g., Hempel and Oppenheim 1948; J.S. Mill 1970/1872: 305). Laws are explanatory, because they show both how the explanandum could have been expected in advance if the relevant conditions would have been known and answer, what Woodward (2000) calls, what-if-things-had-been-different-questions.

However, this philosophical image of laws and explanation does not seem to fit biological generalisations and explanations (Mitchell 2000; Van der Steen and Kamminga 1991; however see Sober 1997, for a defence of the possibility of biological laws). The biological world is diverse and rich in exceptions and biological generalisations have a limited range of explanatory power (Brandon 1997). Moreover, biological generalisations are typically contingent on a specific historical pathway which, depending on past circumstances, could have been different (Beatty 1997). In what follows, these violations will be explicated in more detail.

3.2. *Invariance instead of exceptionlessness*

According to Woodward (2000), the feature that is decisive for generalisations to be explanatory is not its supposedly being exceptionless, but rather

its being *invariant*. Woodward's suggestion concerning invariance is this: a generalisation that describes the relation between two or more variables is invariant if the generalisation does not break down when various conditions change. Some of these other conditions must explicitly figure in the generalisation itself, although a generalisation can be invariant across changes in background conditions as well. It is an important aspect of Woodward's discussion that generalisations may be invariant under *some* changes but need not be invariant under *all* changes. Hence, unlike lawfulness, invariance is not an all-or-nothing matter. Depending on the range of changes over which a generalisation continues to hold, generalisations may be more or less invariant. A generalisation is more invariant if it holds under a larger set of changes or a more important one than other generalisations. Thus, according to Woodward, there is a connection between the range of changes under which a generalisation is invariant and explanatory depth: fragile generalisations do have a lot of exceptions and hold under some but not many changes in conditions, while generalisations that hold under more changes provide deeper explanations.

An example provided by Woodward (2000) of a generalisation in physics is the ideal gas law: $PV/T = R$, describing the relation between the pressure, the volume, and the temperature of a gas, and the constant R. If, e.g., the temperature of the gas is changed, the pressure or the volume of the gas (or both) will change in such a way that R remains constant. Thus the generalisation continues to hold in the face of these changes. That is, the generalisation is invariant under changes in the temperature of the gas. The generalisation, moreover, is also invariant under interventions in background conditions: e.g., the mass of the gas (a variable that does not figure in the generalisation and is thus a background condition) can be changed without the generalisation breaking down. Also, more trivial background conditions like the colour of the container holding the gas can be changed while the generalisation continues to hold. But the ideal gas law is, despite its name, not truly a law, since it is not exceptionless: the generalisation does not hold when the volume of the gas is small in relation to the volume of the constituent molecules. Thus, although the ideal gas law continues to hold under a lot of changes in conditions, it fails under other conditions. Consequently, the ideal gas law should not be considered a law because it is not exceptionless, but it is an invariant generalisation under many changes in conditions and is thus explanatory.

In the natural sciences, generalisations are often equations describing the relation between different variables. In biology on the other hand, explanatory generalisations are often generalisations describing *mechanisms*. Mechanisms are compositions of entities and activities, which are organised in such a

way that they are productive of regular changes from start to finish conditions (Machamer et al. 2000). A common strategy to furnish mechanistic explanations is the decomposition of a system into the entities and activities that constitute the mechanism that brings about the behaviour (Bechtel and Richardson 1993). By decomposing a system into smaller entities, mechanistic explanations are downward looking: behaviour at a higher level of analysis is explained by regularities at a lower level. For example, the transmission of a signal from one neurone to another across the synaptic cleft can be explained by a decomposition of the synapse into generalisations about the behaviour of component parts (terminal buttons releasing neurotransmitters, neurotransmitters diffusing across the cleft, post-synaptic receptors reacting with the neurotransmitters).

Also, in the case of biological generalisations describing mechanisms it is invariance rather than lawfulness which is the important characteristic for explanation (Woodward 2001). These generalisations are invariant, as long as they correctly describe the relation between the start and finish conditions when various (other) conditions change.

3.3. *Distribution instead of universality*

Besides having exceptions, biological generalisations often do not cover all space and time. That is, biological generalisations violate the requirement of universality. The present reading of universality differs somewhat from the standard interpretation of universality. The present sense of universality corresponds to what Schaffner called universality$_1$, which refers to organism scope or 'the extent to which a physiological mechanism will be found in all organisms' (Schaffner 1993: 121). As Darden pointed out, this sense of universality is not limited to organism scope; issues of the scope of applicability also arise above and below the organism level (Darden 1996).

Often mechanisms are the result of a history of selection pressures that may have been different. This idea is called the Evolutionary Contingency Thesis (Beatty 1997). Biological mechanisms are products of evolution, but the past histories of organisms could have been different because of different selective forces which would have resulted in very different mechanisms. According to Beatty, it is because of the evolutionary contingency that we cannot expect a single mechanism to underlie a complete field of biological phenomena. In order for a mechanism to be the universal mechanism underlying a specific phenomenon, it has to be assumed that either this mechanism arose very early in history and has been maintained in all organisms ever since as a homologous trait, or that it arose independently in all organisms and has been maintained ever since as an analogous trait. The first option implies an *extreme phylogenetic conservatism*, the second an *extreme*

parallel evolution.[2] Neither options are likely. Beatty therefore concludes that evolution has not resulted in only one mechanism underlying a specific phenomenon but, instead, in a variety of underlying mechanisms. Hence, biological mechanisms do not cover all space, but only a portion of it.

So, mechanisms are not universal but distributed, based on their evolutionary history. According to Waters (1998) a great deal of the generalisations in biology describe distributions, either implicitly or explicitly. They generalise, for example, about the occurrence of species over geographic regions (e.g., the distribution of elephants over Asia and Africa) or of certain types of species (e.g., marsupials over the continents). Distributions may generalise about characteristics over different taxa (e.g., the property of having wings over birds and insects) or about entities over various parts within individuals (e.g., the distribution of dopaminergic neurones in human brains). Although the variety of forms of these generalisations is wide, they all share an important feature: they are not generalisations about the behaviour of entities but about their occurrence (Waters 1998).

3.4. *Explanation without laws*

The considerations above about invariance and distributions have important consequences for explanations in biology. Laws are not essential for successful explanation: an appeal to invariant generalisations or mechanisms may suffice. That is because explanations involving invariant generalisations exhibit a systematic pattern between the various conditions that can be used in order to demonstrate how the explanandum could have been expected and answer what-if-things-had-been-different-questions (Woodward 2000). The ideal gas law, for instance, provides an explanation of why the pressure of a gas in a container increases to a certain value when its temperature increases by demonstrating how the new pressure could have been expected from the information about the new value of temperature, and what the new pressure would have been if the temperature would not have increased or would have increased to a different value.

Invariant generalisations are not laws, however. Compared to explanation based on laws, mechanistic explanation requires a number of further stipulations. These additional stipulations are directly related to the fact that invariant generalisations are on the one hand not exceptionless and on the other hand not universal. According to Woodward, an invariant generalisation is explanatory only in so far as it correctly describes the relation between the variables operating in it, that is, only within the domain of changes in the conditions under which the generalisation remains invariant. Outside of this *domain of invariance* the generalisation has exceptions and is not explanatory. So, the first stipulation concerns the domain of invari-

ance within which the generalisation has no exceptions and hence within which the generalisation can be used legitimately for explanation. The second condition for successful explanation with non-lawlike generalisations results from the violation of the universality requirement. Invariant generalisation provide predictive power and are explanatory, but only as long as one knows whether the mechanism referred to in the explanation is indeed the mechanism operating (Darden 1996). Protein synthesis is explained by the Central Dogma (DNA → RNA → protein) but only for organisms which use DNA as a template for protein synthesis. Because different mechanisms may underlie the same phenomenon, explanations of that phenomenon need a specification of the scope of the underlying mechanism. Information about whether a certain mechanism accounts for a phenomenon or another one is provided by the distribution of that mechanism. So, the second stipulation for successful mechanistic explanations concerns a specification of the mechanism's distribution.

In short, explanations in biology require invariant generalisations, which often describe mechanisms. Invariant generalisations are explanatory but they are not laws because they are neither exceptionless nor universal. Generalisations are only explanatory within the domain of changes of the conditions under which the generalisation remains invariant and only for those cases where the mechanism described by the generalisation is distributed. Successful explanation, then, not only requires invariant generalisations or mechanisms, but also information about their domain of invariance and information about their distributions.

4. Behaviour genomic and evolutionary explanations

4.1. *The domain of invariance of genomic generalisations*

Explanations provided by behavioural genomics are mechanistic explanations (Barendregt 2003a). In the light of the preceding discussion, an analysis of the generalisations that are used in behavioural genomics reveals that these are very fragile and have a lot of exceptions. Whether or not a specific gene will affect behaviour depends on a complex of other genes and environmental circumstances. That is, genetic generalisations are not invariant under all possible changes in conditions. For example, dietary interventions disrupt the genetic effect of a mutation in the gene *PAH*, the mutation that normally causes symptoms of phenylkotonuria (or PKU) such as severe mental retardation. Thus, the generalisation describing the relation between *PAH* and mental retardation often fails as a result of a change in an environmental condition.

Genomic generalisations are invariant but only within a relatively limited domain specified by other genetic and environmental conditions.

A further complication is that almost nothing is known about the boundaries of the domain of invariance of genomic generalisations. There is little knowledge about, for instance, which environmental circumstances might break down the generalisations describing the relationship between genes and cognition, intelligence, or other behavioural phenotypes. From a genetic perspective it is also very difficult to determine the exact boundaries of the domain of changes in the conditions over which behavioural genomic generalisations are invariant. One reason for this difficulty is the impossibility of experimenting in human behavioural genomics. In order to reveal the limits of a generalisation it is essential to manipulate the conditions in it (Woodward 2000), but in human genomics such manipulations are, inter alia for ethical reasons, not always possible.

The fragility of genome-behaviour relationships has also some practical problems for the research program of behavioural genomics: gene finding strategies are often obstructed because specific genes contribute too little to behaviour to be detected. However, the power of genomic searches can be boosted by the use of so-called *endophenotypes*. Endophenotypes are intermediate traits that directly index the behaviour of interest but are thought to be less removed from the relevant gene action (Almasy and Blangero 2001). For instance, genomic research on general cognitive ability (the 'g' factor) may use behavioural endophenotypes which test specific cognitive abilities, such as cognitive tests for executive functioning, inhibitory control, attention, working memory, etc. Because the endophenotypes are thought to be closer to gene action, it is assumed that it will be relatively easier to identify genes for endophenoptypes than for the overall behaviour. In short, this 'divide-and-conquer' strategy (De Geus and Boomsma 2001: 245) promises that detecting genes for behaviour will be advanced by confining human cognition to specific underlying types.

At this point evolutionary psychology's notion that the mind is massively modular may contribute to behavioural genomics. As mentioned, according to evolutionary psychology psychological processes are seen as solutions to specific adaptive problems that our ancestors had to solve and that affected reproductive success. The functions of the brain and other neurophysiological bases are supposed to have evolved in a functionally specialised way. This feature, known as the *domain specificity* of the human mind, results in the hypothesis that the mind has a large number of so-called 'content-rich expert systems' (Duchaine et al. 2001) containing assumptions, favourite expectations and special inference procedures or heuristics to deal with the information and operations the module is sensitive for or prone to

(Gigerenzer and Hug 1992; Gigerenzer and Todd 1999; Gigerenzer 2001). Among the specialised systems suggested by evolutionary psychologists are such modules as cheating detection (Cosmides 1989; Gigerenzer and Hug 1992), face recognition (De Haan 2001), emotion recognition in faces (Van Honk et al. 2001), disgust (Rozin et al. 1986; Rozin and Fallon 1987; Rozin et al. 1993), food aversion (Garcia et al. 1955; Garcia and Koelling 1966; Garcia et al. 1977; Garcia 1981), etc.

As an illustration of how evolutionary psychologists carve psychological phenomena at its evolutionary joints, let us briefly consider the example of semantic knowledge. Research in evolutionary psychology has indicated that semantic memory is divided in two systems: one for assessing semantic information about animate objects and one for inanimate objects (Duchaine et al. 2001). These categories represent 'evolutionarily adapted domain-specific knowledge systems' subserved by distinct neural mechanisms (Caramazza and Shelton 1998). Further distinctions are also suggested, for instance between edible and inedible things. This way, a fine-grained categorisation of psychological mechanisms is developed based on evolutionary thought.

Behavioural genomics may benefit from these finer grained evolutionary categorisations because they may be genetically relevant. The different modules are thought to have evolved under selection of different (compositions of) genes. So, it may be expected that the modularity of the behaviour categorised by evolutionary psychology somehow maps onto a genomic organisation. In other words, the way that evolutionary psychology describes the human cognitive architecture should provide hints at the organisation of the genomic background. Moreover, modules are domain specific: they are highly specialised mechanisms serving very specific tasks. They have been selected because they produce, in a regular fashion, adaptive behaviour, but they do so only within a domain of a specific kind of content. Changing the content outside the evolutionary context renders the module irrelevant or even misleading. That means that the generalisations describing the relationships between modules and behaviour remain invariant but only within the domain to which the module is an adaptation. If it is indeed true that different genes underlie different modules, it follows that the generalisations describing gene-behaviour relationships will be likewise domain specific: the domain of invariance of gene-behaviour generalisations will probably correspond to a more or lesser extent to the domain specificity of the adaptive solutions those genes have been selected for. Thus, the boundaries of the domain of invariance of genomic generalisations may be brought to light by investigating the domain-specificity of the adaptive solutions those genes have been selected for. This way, evolutionary psychology not only provides

hints at the genomic organisation, but also helps to describe the domain of changes under which genomic generalisations are invariant.

This line of reasoning also leads to a more practical recommendation concerning the choice of endophenotypes. At present, evolutionary considerations are not among the criteria for useful endophenotypes (see, e.g., De Geus and Boomsma 2001). But if the organisation of the genome corresponds to the adaptive organisation of the human mind and if gene-adaptation relationships are less fragile than more general gene-behaviour relationships, it might be predicted that genes for adaptations will be relatively easier to detect than genes fort traits which are not adaptations. This suggests that the endophenotype approach to behavioural genomics can be improved by focussing on *adaptive* endophenotypes. Behavioural genomics may try to make the way they 'divide and conquer' human behaviour in agreement with the way evolutionists carve behaviour at its evolutionary joints. Besides choosing endophenotypes like inhibitory control and executive functioning, choosing evolutionary relevant endophenotypes can thus be a further advance for behavioural genomic research.

A suggestion which originated in linguistics and has been adopted by evolutionary psychologists provides another, more detailed example of where evolutionary considerations contribute to behavioural genomics (Wimsatt 1986; but see also his rethought and clarified analysis in Wimsatt 1999). The concept of a Universal Grammar refers to a set of basic principles that humans possess at birth to develop the production and perception of linguistic utterances, the grammar of which is constrained to certain sets of rules and combinations of rules (cf. e.g., Chomsky 2000; Jackendoff 2002; Pinker 1999). Theories concerning language acquisition suppose that there is a genetic disposition (G_i) to produce a basic set of grammatical rules. These rules, however, can take different forms, e.g., X can take the form of X_1 or X_2, depending on an environmental input. An example of such grammatical rule is the so-called PRO-drop rule which allows the speaker to omit the subject in a sentence (Pinker 1995). This rule is set to 'on' in Spanish (which is a null-subject language where one can say 'goes to the beach' without a subject) whereas the rule is set to 'off' in English (where one cannot say the equivalent). In the child's grammar the PRO-drop rule is set to 'on' or 'off' depending on the input (e.g., English or Spanish). Other, more subtle linguistic rules (Y_1, Y_2, Y_3) will depend on whether X takes the form of X_1 and X_2. For instance, once X is set to X_1, Y_3 is no longer possible (though $X_2 \rightarrow Y_3$ still is possible), which leaves the environment open to influence the decision between Y_1 and Y_2 under the condition of X_1. In short there are genetic sources for the variations $\{G \rightarrow X_1 \rightarrow Y_1\}$, $\{G \rightarrow X_1 \rightarrow Y_2\}$, $\{G \rightarrow X_2 \rightarrow Y_3\}$, which depend on the environment to be expressed in one way or

the other. In a case like this evolutionary considerations could come up with analyses that lead to predictions about the organisation of the genome, which probably could not have been made on the basis of behavioural genomics alone. Yet, it remains a matter of empirical investigation to find out whether there is one gene (or combination of genes) that is responsible for the $X_i \rightarrow Y_j$ sequence, or whether X and Y are under the influence of different genes.

4.2. *The distribution of genomic generalisations*

Besides exceptions, genomic generalisations typically lack universality. There is often more than one genetic mechanism underlying the same (behavioural) phenomenon. Information about the distribution of genes and genetic mechanisms is therefore an essential part of genomic explaining. What is needed is a search within a population for the genes involved in the generalisation. It is, inter alia, important for the significance of a finding: even in the case that a gene is without exception followed by a specific trait, if the gene is very rare while other causes are more common, it explains relatively little.[3] An example is the gene *BRCA1*. Individuals with a mutation in *BRCA1* have a very high risk for developing breast cancer (60–80%), but the gene is relatively rare and explains about 3–7% of all breast cancers (Bishop 1999; Keen and Davidson, 2003). Another behavioural example is Creutzfeldt Jacob's Disease (CJD) which is associated with the *PRNP* gene: a mutation in *PRNP* invariantly causes CJD, but the gene is responsible for only 10–15% of all cases of CJD (DeArmond and Bouzamondo 2002).

Although genetic researchers are able to estimate and calculate the distribution of genes, what is usually lacking is an explanation or an account of these distributions. What is missing is a higher level perspective which explains why the distributions are as they are. Behavioural genomics does not systematically account for the way genes are distributed, e.g., among different populations or geographical regions or other environments. It remains a mere description of the scattering of genetic mutations within a more or less well defined class of subjects.

Evolutionary psychology may contribute to an account of the distributions of genes since the latter results from the inheritance of the genes from parent to offspring. Especially individuals that are well adapted to their environment are more likely to transmit the genes they carry and, as a result, those genes whose effects enhance the reproductive fitness of the individual in a certain environment are probably more common there. So, a particular distribution of genes is the result of the history of the adaptive consequences of the genes involved. Thus, knowledge of the adaptive features of human behaviour and their functions in the adaptations in the ages of their original appearance as a feature may be of help for a systematic description of genetic distribu-

tions. Gene-adaptation relationships can be supplemented with knowledge of adaptation-environment relationships as revealed by evolutionary psychology. The latter relationship may suggest in which environments the genes are more likely distributed, and, more importantly, suggests an explanation of why these distributions are as they are. Thus, a second way that an adaptive account may contribute to behavioural genomics is by accounting for the genetic distributions that are relevant for genomic generalisations.

An example is the already mentioned disease PKU (Barendregt 2003a). This genetic mutation causing the disease is relatively uncommon, but more frequent in countries like West-Scotland and Ireland. The distribution of the mutation can be explained by an adaptive account (Woolf 1986). The mutation results in a mutant enzyme which provides some protection against a specific toxin, viz. Ochratoxin A. Due to this protection, heterozygous pregnant women (who are themselves not affected by the disease because PKU is a recessive disorder) have fewer spontanious abortions than others. Ochratoxin A is produced by moulds that prefer to grow in mild and wet climates. Combined with the fact that West-Scotland and Ireland suffered serious famines in the past which makes it more probable that mouldy grain is consumed, this explains why this gene is relatively frequent in those regions.

To avoid misunderstanding, the present authors do not recommend evolutionary psychology to completely abandon their focus on mechanisms which are universal in the human species. The point is that if adaptive genetic variation is also taken into account, the research strategy of evolutionary psychology might become relevant for behavioural genomics. Evolutionary psychology does not need to radically change their strategy: evolutionary psychologists already recognise that genetic origins of behaviour leave room for variation of two kinds. If a certain gene (or combination of genes) produces a strong tendency to develop a certain behaviour in a standard way, room for variation in the expression remains (i.e., due to genetic room within constraints) as well as room for variation in the expressed behaviour that is due to environmental influences on the behaviour.

5. Conclusion

The view proposed in this paper is limited in several respects. First of all, we have only discussed the possible relevance of an adaptive account for behavioural genomics and not visa versa. Also, it was asserted that, because behavioural genomics is about individual variants, only adaptive genetic variation provides points of contact and not pan-human uniform adaptations. Recent developments in behavioural genomics, however, suggest a more complicated picture. Although behavioural genomics emphasises individual

differences, its account may extend beyond variance and throw light on behavioural uniformity as well. This is a result of the shift from quantitative to molecular analyses, which brings with it a conceptual shift from genes as an *abstract* statistical factor for individual differences to genes as *physical* entities with 'real' causal powers. While quantitative analyses remain silent on the exact relation between genes and phenotypes, molecular studies try to open this black box by targeting the causal paths that lead from genes to behaviour aiming at knowledge about the intervening mechanisms by which different genes bring about different behavioural outcomes (Plomin and Essi 2001). Such knowledge not only includes behavioural variants but is also advantageous to behavioural research focusing on species universals: laying bare the intervening mechanisms provides a basis for understanding the functional anatomy of neurones and brain parts and their role in behavioural performance (De Geus and Boomsma 2001). Consequently, adaptive explanations not only complement explanations of individual variation, but they may, in turn, also *benefit* from genomic explanations. Hence, the relationship might also work the other way round. Another consequence is that the overlap between evolutionary psychology and behavioural genomics might include more than just adaptive genetic variation: adaptive uniformity might also be a shared domain of interest.

Secondly, we have focused on the unification of adaptive and genetic explanations as *a conjunction*, that is, in certain situations genetic explanations can be amplified by adaptive explanations. What has not been examined is whether one approach may be reduced to the other or whether the two may dissolve into a third theory. The shift from statistical to molecular analyses might provide grounds for a stronger kind of unification than mere conjunction. For instance, a molecularly based behavioural genetics raises the question whether the *reduction* relation might be applicable between evolutionary psychology and behavioural genomics. These questions are similar to the older debate in the philosophy of biology on the reduction of functional biology to molecular biology (see e.g. Rosenberg 1997; Schaffner 1993: chapter 9). The outcome of that debate seems to be an antireductionist consensus partly based on the multiple realisability argument (Hull 1974; Kitcher 1984; Sober 1999). If we may extend those ideas to the relation between evolutionary psychology and behavioural genomics, a preliminary conclusion might be that reduction may prove not to be the appropriate kind of unification there.

Finally, we have not criticised the foundations or assumptions of either behavioural genomics or evolutionary psychology. But both approaches have not gone unchallenged (e.g., Gottlieb 1998; Looren de Jong and Van der Steen 1998). For example, the thesis that the human mind is massively modular has

been contested (e.g., Buller and Hardcastle 2000), which is pertinent in the context of this paper, because if the modularity of the mind has to be rejected one of our proposals why evolutionary concerns may be relevant for behavioural genomics would be undermined. Of course, it might turn out that one of the two traditions (or both) is essentially based on mistaken assumptions. But, if that would be the case there would be no question any more whether one might contribute to the other. It should be kept in mind that it is not our aim to scrutinise the assumptions of the two biological research programs in psychology but to investigate the possibility of unification-relationships.

Taking these limitations into account, we conclude that evolutionary psychology and behavioural genomics need not be considered separate disciplines. They need not be disconnected islands between which no communication is possible. The areas of both disciplines partly overlap, at least where genetic variation is adaptive. In these overlapping areas points of contact exist. We have proposed two such points of contact where evolutionary psychology might be relevant for genomic explanations. Because of their lack of lawfulness, biological explanations require an account of the domain of invariance of their generalisations and an account of the distribution of the mechanisms. These accounts may be provided by an adaptive perspective.

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Notes

[1] What it generally means exactly for two theories to be mutually consistent is not discussed. Apparent inconsistencies, however, suffice in any concrete case to block attempts for unification and should be resolved firstly.

[2] One example of a principle that might be extremely phylogenetically conservative is the genetic code. The code seems to be universal for all organisms, which is explained by Crick by the fact that once the code had been established any changes in it would have had enormous and detrimental effects. Many researchers, however, think that the genetic code is still evolving and hence not universal.

[3] John Beatty has developed the idea that the non-universality of biological theories and the occurrence of relative significance disputes in the biological discipline are related phenomena (Beatty 1997).

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